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Monoarticular Arthritis Update: Current Evidence For Diagnosis And Treatment In The Emergency Department

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CME Objectives

Upon completion of this article, you should be able to:

1. Identify the most common causes for ED monoarticular arthritis presentations.
2. Discuss the value of serum blood tests and arthrocentesis in diagnosing septic arthritis.
3. Discuss current therapies for acute gout and septic arthritis.

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Abstract

Monoarticular arthritis presentations in the emergency department are increasing as the population ages and gets heavier. Many etiologies — from trauma to infection to autoimmune-mediated inflammation — are associated with significant disability or early mortality, and their treatments are associated with adverse effects. A systematic approach to evaluating patients with monoarticular arthritic complaints is important for relieving pain, diagnosing systemic illness, and unmasking true arthritis emergencies. Septic arthritis is a rapidly destructive process that can cause significant disability in a matter of hours or days, with relatively high mortality. Other causes of monoarticular arthritis may cause disability in the long term. In all cases, accurate diagnosis and appropriate therapies are crucial for resuming activities and preventing long-term deficits. This review examines the diagnosis and treatment of monoarticular arthritis, with a focus on recent evidence in the diagnosis of septic arthritis and new research on gout therapies. Modalities for pain control and new techniques for imaging are discussed.

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Case Presentations

A middle-aged man with diabetes and hypertension has been waiting patiently to be seen, with a complaint of right knee pain and swelling that has gradually progressed over several hours. There is no history of trauma, recent or remote, but he describes several gout-like episodes of pain in both feet in recent years, which improved with rest and NSAIDs. He was triaged as an ESI4, and the waiting room is packed. Fortunately, the charge nurse comments on how uncomfortable he looks and he is brought into the ED for evaluation. The patient is afebrile, but the knee is hot, beefy red, and swollen. The ED is over-capacity and the patient's history is reassuring, so you consider keeping him in a chair. But that knee looks impressive and you wonder if your plan is aggressive enough.

Just then, your nurse brings a new patient back to the adjacent room. He is loudly complaining of shoulder pain, a flare-up resulting from a shoulder injury years before. He is 40 and has no other medical problems. "My pain doc usually just gives me a shot, but he's out of town," he said. You order a film and 800 mg PO ibuprofen, but you wonder if an intra-articular injection of analgesia and steroids would be better.

Introduction

Arthritis is the most common cause of disability in the United States,[1] and this is likely to increase as the population gets older and heavier. Because of the limitations in activity that are associated with joint inflammation, patients with arthritis complaints frequently present to the emergency department (ED).[2]

Monoarticular arthritis, in particular, represents a diagnostic dilemma. Presentations run the gamut from exacerbations of chronic osteoarthritis (OA) that has been developing for years to rapidly progressive infectious arthritides that can quickly disable or kill in days. Identifying and promptly treating septic arthritis, a true medical emergency, remains the cornerstone of the workup for monoarticular arthritis. Both inside and outside of emergency medicine, misconceptions in the diagnosis of septic arthritis persist. New evidence about the limited role of examination, imaging, and blood testing, as well as the importance of arthrocentesis, are covered in this review.

Beyond septic arthritis, other causes of monoarticular arthritis can still be painful and debilitating. New data and guidelines for therapy related to the diagnosis and treatment of gout, pseudogout, and OA are reviewed as well as diseases that can occasionally present as monoarticular arthritis, such as Lyme disease and reactive arthritis.

Critical Appraisal Of The Literature

Recent years have seen renewed interest in emergency arthritis presentations and management, particularly surrounding diagnosis of septic arthritis and treatment of gout. There are also new guidelines from The European League Against Rheumatism (EULAR), the American College of Radiology (ACR), and the American Academy of Orthopedic Surgeons (AAOS) as well as Cochrane Library of Systematic Reviews concerning arthritis management. These guidelines have emphasized evidence-based recommendations, when possible, although the priority is often on long-term management, and definitions of "acute" and "short-term" may seem unusual to emergency clinicians.

Ovid MEDLINE® was queried for clinical trials since 2004 that matched *emergency* and *arthritis.* The website www.guidelines.gov was also queried using similar search terms over that period. The Cochrane Database of Systematic Reviews was surveyed for applicable systematic reviews. Finally, the recent orthopedics and rheumatology literature was searched regarding new therapies for gout and other monoarticular arthritides.

Epidemiology, Etiology, And Pathophysiology

The body's diarthrotic (moving) joints are composed of 2 ends of subchondral bone capped with articular cartilage. Cartilage is avascular, aneural tissue composed of a matrix of collagen fibers and proteoglycans capable of tremendous load bearing. Joints are surrounded by a capsule lined with a thin synovial membrane.[3] Synovial fluid within joints is an ultrafiltrate of blood supplemented with hyaluronic acid and proteins, making a viscous, lubricating synovial fluid that enables near-frictionless joint movement.

Arthritis can be triggered by trauma, infection and its response, or inflammation from other causes such as crystal deposition or autoimmune processes.[4-6] The incidence of septic arthritis in the general population is bimodal, with a peak for young children and adults over age 55,[7] and has been approximated at 2 to 10 cases per 100,000, per year. Gonococcal arthritis, the most common infective arthritis in the United States in the 1970s and 1980s, has experienced a decline, though it remains the most common joint infection among the sexually active population. There is a 4:1 female-to-male predominance, and pregnancy and menstruation are risk factors for dissemination.

Gout prevalence is much higher, with 2.7% of the United States population estimated to have experienced symptoms.[8] Risk factors include obesity, hypertension, diabetes, thiazide and cyclosporine use, and radiocontrast exposure. Flares can be brought about by excess alcohol, stressors from illness,

trauma or surgery, and purine-rich diets.[9] Coffee has been shown to decrease risk.[9]

OA is the most common form of arthritis among adults, with 50% of the United States population exhibiting signs of the disease by age 65 and near universal evidence of the disease (if not necessarily symptoms) by age 75.[10] Obesity increases the likelihood of OA in the lower extremities.

Lyme arthritis is often considered a polyarticular arthritis, though asymmetrical. After the period of early disseminated disease, if untreated, 50% to 60% of patients develop asymmetric arthritis within 6 months, most commonly in large joints such as the knee.[11] Over years, even without treatment, episodes of arthritis typically abate in frequency and intensity. Lyme disease incidence in the United States is currently estimated at 25,000 cases per year.[12]

In the ED, trauma is the most common cause of acute monoarticular joint swelling and pain.[13]

Prehospital Care

Monoarticular arthritis care in the prehospital setting — especially in cases of trauma — is centered around providing comfort in transport to the ED for further evaluation. Extremity or joint immobilization is the mainstay of prehospital management, though analgesia can also be provided depending on local protocols and patient characteristics.

Emergency Department Evaluation

History

While important in determining the scope of the workup that comes later, the history and physical examination have a limited evidence base from which to draw. The cardinal features in the history include onset, duration, chronicity, and modifying factors. These features may help distinguish, for instance, classic presentations of OA — which progresses during the day and with use — from rheumatoid arthritis (RA), which starts as a stiffness in the morning and improves with time. A presentation of gout may become clearer if there is a history of renal stones, thiazide diuretic use, or a diet of meats and shellfish, beer, and high-fructose corn syrup. Recent camping or travel to endemic areas may be the only historical key in diagnosing Lyme arthritis. Similarly, for reactive arthritis, a history of dysentery or urethritis in the weeks prior may help establish the diagnosis. A history of repetitive stress or mild trauma to a joint may favor bursitis over true arthritis.

Physical Examination

The first goal of the physical examination is to distinguish between a true arthritis and periarticular inflammation such as from a bursitis, tendinitis, or localized cellulitis. The tenderness and swelling of periarticular inflammation is not uniform across the joint; pain is most commonly elicited during active contraction or passive stretching of focal muscles and tendons. In contrast, true arthritis is painful with both active and passive motion, due to the inflamed synovium lining the entire joint capsule. The redness, tenderness, warmth, and swelling are more generalized.

Skin findings may shed light on a variety of arthritides: tophi are seen in gout, and Bouchard nodes on the proximal interphalangeal (PIP) joint and Heberden nodes on the distal interphalangeal (DIP) joints are seen in OA. Reactive arthritis may be clarified by the presence of mucocutaneous ulcers, conjunctivitis, balanitis, or the sausage-like fingers similar to psoriatic arthritis. Gonococcal arthritis may be associated with pustular lesions seen elsewhere in disseminated infections.

A systematic review of the physical findings suggestive of septic arthritis reported a low sensitivity for fever; indeed, the presence of fever actually decreases the likelihood of septic arthritis. (Likelihood ratio [LR] 0.67, 95% confidence interval [CI], 0.43-1.00.)[14] Tenderness, palpation, and range of motion have not been sufficiently studied to the point where firm conclusions can be drawn about their utility.

Serum Laboratory Values

Serum blood testing is of only modest utility in the ED workup of monoarticular arthritis. There is no general screening test to help distinguish, for instance, autoimmune or RA from other forms of arthritis. Erythrocyte sedimentation rate and C-reactive protein may have some value in diagnosing polymyalgia rheumatica and other specific forms of arthritis.[15] Their value for septic arthritis is minimal and discussed in following sections. Similarly, serum uric acid can be misleading in the workup of gout and should not be used to guide decision-making in the ED.

Radiology

There is little evidence to support the utility of obtaining x-rays in the setting of acute monoarticular arthritis in the ED. Plain films may be more helpful in patients with chronic disease, although because acute presentations are so undifferentiated, it is common practice to search for various signs of specific arthritides such as chondrocalcinosis or overhanging margins, joint space narrowing or osteophytes, or fractures (pathologic or traumatic). At the very least, there are recommendations that plain films can be obtained for baseline imaging, for future reference.[16]

Magnetic resonance imaging has demonstrated utility at diagnosing tendinous and ligamentous disruption at the shoulder and knee and early edema in periarticular structures, and it is more sensitive than x-ray in diagnosing early osteomyelitis.[16-18]

Bedside ultrasound has been suggested as a tool to facilitate difficult joint aspirations. In a study of patients referred to a rheumatology clinic, ultrasound-guided needle placement succeeded in 31 of 32 arthrocentesis attempts, compared to 10 out of 32 nonultrasound-guided controls.[19] Additionally, ED ultrasound may help identify gout and help distinguish gout from pseudogout. **(See the Controversies And Cutting Edge section, page 15.)**

Arthrocentesis

Emergency indications for arthrocentesis in the workup of joint pain include obtaining joint fluid for analysis, draining tense hemarthroses in patients with trauma, evaluating whether a laceration communicates with the joint space, and instilling analgesics and anti-inflammatory agents for the treatment of acute and chronic arthritis. The American College of Rheumatology Guidelines Committee recommends arthrocentesis on patients with an established history of arthritis who present with fever and new joint pain or effusion.[20] Although there is scant literature on the true incidence of inoculated joint spaces, emergency arthrocentesis through overlying cellulitis is relatively contraindicated. Avoiding the infected area during the puncture is recommended, if possible; however, if septic arthritis is in the differential, arthrocentesis should be performed. Coagulopathy is another relative contraindication, though arthrocentesis in patients with an international normalized ratio (INR) as high as 4.5 has been performed without hemarthrosis or soft-tissue hemorrhage, using an 18-gauge needle for knees, 20-gauge needle for most other joints, and a 25-gauge needle for metatarsophalangeal (MTP) joints.[21]

Complications Of Arthrocentesis

The primary complications of arthrocentesis are bleeding or infection in the joint space, allergic reaction to anesthetic agents, and long-term corticosteroid-related complications. Dry taps (when no fluid is aspirated after joint puncture) are more common in patients with chronic arthritis owing to obstructing tophi or anatomic abnormalities in the synovium and periarticular tissues. Using a smaller syringe or a larger needle may help in such cases. Many laboratories will accept just a single drop of synovial fluid for use in crystal analysis on a slide mount.

Arthrocentesis Technique

A successful tap of a joint begins with cushioning the joint of interest, making the patient comfortable, exposing the joint, and allowing adequate flexion, all of which will maximize the chance of first-pass success. Muscle tension during the procedure can reduce the synovial volume, making the procedure more difficult. Carefully palpate the bony landmarks and prepare the skin using an aseptic technique. Adequate local anesthesia can be achieved either by use of a vapor coolant or by local infiltration with anesthetic solution such as 1% or 2% lidocaine. Using an 18- or 19-gauge needle (for large joints) or 20- to 25-gauge needle (for smaller joints) attached to a syringe, puncture and aspirate the joint space while taking care to avoid abrasion of the articular cartilage. Excessive suction may bring synovial tissue or blood into the needle, limiting aspiration or confounding analysis, respectively. **See Table 1** for specific landmarks and positioning for various common joints aspirated in the ED.

After aspiration, a long-acting anesthetic can be instilled to alleviate pain, though evidence supporting this practice in the ED is limited, and some in vitro models suggest bupivacaine is chondrotoxic.[22,23] Instilling steroids can be considered if septic arthritis is not a concern.

Synovial Fluid Examination

Bedside evaluation of the color, clarity, and viscosity of synovial fluid can aid in making the diagnosis. Normal synovial fluid is clear, colorless, and has a viscosity that permits stretching of a "string" of fluid between the thumb and forefinger. Inflamed fluid is more opaque, from elevated white blood cells (WBCs), and has a more watery viscosity due to enzymatic breakdown of glycosaminoglycans. In a prospective study of 80 synovial fluid samples, rheumatologists were able to distinguish inflammatory arthritides by appearance alone, with 94% sensitivity and 58% specificity.[24]

Differential Diagnosis And Treatment

Acute Joint Pain

After an appropriate history and pertinent physical examination have been obtained, the differential for acute joint pain is already narrowed. Laboratory testing and imaging will further facilitate an accurate assessment and treatment plan. Traditionally, joint pain has been classified into monoarticular and polyarticular categories, but given that multiple diagnoses may fall into both categories, such a classification can be misleading.

Septic Arthritis

Septic arthritis is the true joint emergency that no provider can miss; it threatens life and limb. It is the feared diagnosis, particularly when someone presents with singular joint pain. This process affects 2 to 10 persons per 100,000 annually in the United States, especially young children and those older than 55 years.[7] Other risk factors that prompt suspicion for an infected joint include intravenous (IV) drug use, alcoholism, diabetes, immunocompromised states, chronic arthritis, low socioeconomic status, and history of a prosthetic joint. Missing the diagnosis of septic arthritis can result in the development of osteomyelitis, cartilage destruction and subsequent joint replacement, or generalized sepsis.

Table 1. Arthrocentesis Techniques[25-27]

Wrist: Radiocarpal Joint (Dorsal Approach)

1. Identify landmarks by palpating the Lister tubercle at the distal end of the dorsal radius.
2. Palpate the extensor pollicis longus tendon, which passes over the radial side of the Lister tubercle (best palpated while the wrist is in extension). You will insert the needle on the ulnar side of the EPL tendon, just distal to the Lister tubercle.
3. Lay the wrist on a cushion so that it is flexed 20° to 30°.
4. Apply traction from the fingers and mild ulnar deviation, and insert a 22-gauge needle dorsally.

Elbow: Radiohumeral Joint (Lateral Approach)

1. Identify landmarks by extending the elbow and then palpating the depression between the lateral epicondyle of the humerus and the head of the radius.
2. Keeping your finger on the radial head, flex the patient's elbow, pronate the forearm, and lay the palm on a flat surface.
3. Insert a 20-gauge needle just distal to the lateral epicondyle, directed medially.

Shoulder: Glenohumeral Joint (Posterior Approach)

1. Lay the patient's arm, internally rotated, across the waist.
2. Identify the posterolateral corner of the acromion.
3. Insert a 20-gauge needle 2 cm to 3 cm inferior to this point, directed anteriorly and medially (and slightly superiorly) toward the coracoid process.

Hip: Acetabulofemoral Joint (Lateral Approach)

1. Lay the patient supine and internally rotate the affected leg.
2. Palpate the greater trochanter.
3. Insert a 3.5-in 18-gauge needle superiorly to the trochanter, horizontal and parallel to the stretcher. If the femoral neck is encountered, withdraw 2 mm to 4 mm and redirect slightly cephalad until synovium is aspirated.

Knee: Patellofemoral Joint (Medial Approach)

1. Flex the knee 15° to 20° (often achieved with a rolled towel under the knee). The foot should be perpendicular to the floor.
2. Palpate the anteromedial patellar edge at the patellar midpoint or superior portion.
3. Insert an 18-gauge needle 1 cm medial to this point, directed toward the posterior surface of the patella.

Ankle: Tibiotalar Joint (Anteromedial Approach)

1. With the patient supine, have the patient plantarflex the foot.
2. Identify the anterior tibial tendon.
3. Insert a 3.5-in 20- or 22-gauge needle medial to this tendon, in the depression at the anterior edge of the medial malleolus.

Metatarsophalangeal Joint (Dorsomedial Approach)

1. Identify the distal metatarsal head and the proximal base of the first phalanx.
2. Identify the extensor tendon by asking the patient to extend the great toe.
3. While the patient is supine, flex the toe 15° to 20°, and then apply traction.
4. Insert a 22-gauge needle dorsally, just medial to the extensor tendon.

Abbreviation: EPL, extensor pollicis longus.

The classic presentation of the septic joint is fever with joint pain and effusion, generally in a large joint. Providers may naturally look for decreased range of motion or tenderness, but there is insufficient evidence to rate the prognostic value of these signs. A history of subjective fever has a wide range of sensitivity, from 44% to 97%.[28] One study reported a 90% sensitivity of fever in the first 48 hours of presentation, but temperature upon presentation was not stated.[29] Perhaps a more clinically useful way for considering signs and symptoms of septic arthritis is to think of which special population the presenting patient may fall into.

In the sexually active population, gonococcal arthritis must always be considered, as it is the most common form of joint infection in this group. In 2010, there were 309,341 cases of gonococcal arthritis in the United States.[30,31] Those at even higher risk are pregnant or menstruating women and/or patients who have a complement deficiency.[31-34] While gonococcal arthritis is less likely to yield long-term joint pathology, it has a unique clinical presentation and thus providers must specifically consider it in order to correctly identify it.[35] More commonly, gonococcal arthritis affects a few joints – usually the wrist, knee, or ankle – rather than just 1 joint. Furthermore, it may manifest in conjunction with tenosynovitis, rash, migratory arthralgias, and/or bacteremia.[31,36]

Another special population to consider is the immunocompromised. Septic joints in this patient category may not present classically; they may be afebrile or have minimal, if any, effusion. Consider the pathophysiology that is required to mount a fever or develop an effusion: in a person with a healthy immune system, the presence of bacteria in a joint promotes an inflammatory cascade that results in the enzymatic, cellular, and cytokine breakdown of articular cartilage. In those with low CD4 counts or in the elderly, for example, such a cascade may not occur at the local joint level, and an effusion or fever may be absent or unimpressive to the examiner. Mechanical joints can also become infected, either from direct inoculation secondary to the surgery itself or via hematogenous spread. Because the joint is prosthetic, the native immune response of the host is diminished, as is the bioavailability of therapeutic antibiotics.

Given the wide range of possible presentations of the septic joint and the massive implications of mismanagement, appropriate investigation and interpretation of results are crucial. Having established that the history and physical examination cannot reliably exclude the diagnosis, especially in certain populations, providers must rely on diagnostic testing. Commonly drawn (and requested by consultants) blood work includes a complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Nonetheless, retrospective and prospective trials have shown that there are

no values for these serum markers for which one could rule in or rule out septic arthritis. For serum WBC count < 10,000, for ESR < 30, and for CRP < 100 mg/L, the -LRs for septic arthritis were 0.28, 0.17, and 0.44, respectively, but with wide 95% CIs that rendered the tests useless for changing pre-tap probability for septic arthritis.[14,28,37]

Similarly, plain films of the joint will not remove the possibility of the feared diagnosis, as bony changes in septic joints take place over a long period of time rather than at first presentation. Rather, radiographs can help in the evaluation for osteomyelitis and to establish a baseline for the patient. **(See Figure 1.)**

The gold standard for diagnosis of a septic joint is a positive culture of synovial fluid, but this can take days, and is thus not helpful in emergency practice. Consequently, management decisions must rely on clinical acumen and laboratory results. The emergency clinician's conundrum is that gestalt is imperfect, and numerous studies have revealed the flaw in using serum levels to rule in or out septic arthritis as a diagnosis. Traditionally, glucose and protein levels of the synovial fluids are requested, but these studies are not helpful in diagnosing or ruling out septic arthritis. Synovial glucose levels have failed to significantly increase or decrease post-test probability for a septic joint; synovial protein levels < 30 g/L have a -LR of just 1.1.[28]

The most important tests to send are Gram stain and aerobic and anaerobic cultures of synovial fluid. The Gram stain will yield bacteria in 50% to 80% of infected joints, and it helps narrow the appropriate antibiotic regimen while the patient is still in the ED. Even a negative Gram stain can be misleading, particularly if the appropriate plating is not requested by the provider. In the sexually active population, gonorrhea is of particular concern, and yet cultures of synovium and blood are positive for *Neisseria gonorrhoeae* in only 10% to 50% of true gonococcal septic arthritis cases.[35] This yield can be improved if the provider requests specimens to be plated on Thayer-Martin medium or evaluated via polymerase chain reaction (PCR). Direct communication with laboratory personnel, thus recruiting them into the diagnostic strategy, can positively impact patient outcomes. Also, obtaining cultures of the cervix or urethra, rectum, and pharynx of patients may be instrumental to making the diagnosis, as cultures may be positive in 75% of gonococcal arthritis cases.[32]

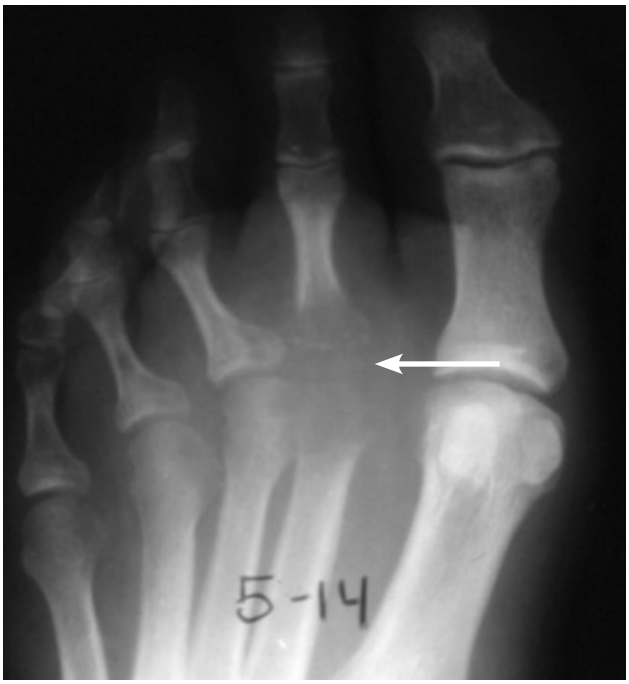
There is some literature to suggest a potential role for serum procalcitonin, tumor necrosis factor, and/or cytokine levels, but practically, such test results are not available in a timely manner for the emergency clinician. The answer is in the synovial fluid: synovial WBC (sWBC), synovial lactate dehydrogenase (sLDH), and synovial lactate (sLactate) are more likely to point providers in the right direction while awaiting Gram stain and culture results.[28] **(See Table 2.)** The higher the WBC count of the synovial fluid, the harder it is to rule out septic arthritis as a diagnosis. If the sWBC count is > 25,000/mm^3, the +LR is 3.2. This +LR rises with sWBC; at > 100,000/mm^3, an aggregate +LR from studies was 13.2 (95% CI, 3.6-51.1), with 2 studies suggesting infinite likelihood.[38,39]

The merit of using sLDH to determine the likelihood of septic arthritis was demonstrated in a single trial.[38] sLDH of > 250 U/L demonstrated a +LR of 2.0. In a systematic review of 4 trials, sLactate of 5.6 mmol/L or greater was shown to have a +LR of 2.4, with other studies suggesting values > 10 mmol/L approaching infinite likelihood.[28] In comparison, a serum ESR of > 30 mm/hr may have a +LR of only 1.3. In one study, PCR showed promise for revealing organism-specific results within 3 hours, but more evidence and availability are needed to make this a worthwhile test from the ED.[40]

The threshold for diagnosing septic arthritis based on sWBC and synovial polymorphonuclear (sPMN) cell count is lower in those with a prosthetic joint. In such cases, an sWBC count > 1100/mm^3 or having more than 64% sPMN cells is sensitive and specific for infection.

In immunocompromised patients (including those with CD4 counts < 200/mm^3) and in the elderly, consider the possibility of mycobacterial or fungal arthritis.[41] Synovial fluid should specifically

Figure 1. X-Ray Findings In A Septic MTP Joint



Arrow points to poorly defined, bony erosion in the second MTP joint.
Abbreviation: MTP, metatarsophalangeal.
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be sent for examination in search of these entities. Tuberculous arthritis should also be considered in populations at greater risk, including prison inmates and immigrants or those working or living in close proximity to these populations.

Management Of Septic Arthritis

Management of the patient with a septic joint is two-fold: The provider's most important role is the reduction of morbidity and mortality. To that end, facilitating definitive care and early and appropriate institution of antibiotics are paramount. There are no randomized controlled trials regarding antibiotic regimens in septic arthritis; the choices are based upon suspicion for particular organisms. Vancomycin 30 mg/kg IV twice daily is appropriate for Gram-positive coverage, including methicillin-resistant *Staphylococcus aureus* (MRSA). It is difficult to predict which patients will be affected by MRSA rather than methicillin-sensitive *S aureus* (MSSA), but neonates and the elderly are particularly at risk, and the associated MRSA morbidity and mortality is worse if there is delay in treatment or inappropriate regimens are selected.[42,43] For Gram-negative bacilli, third-generation cephalosporins are appropriate. Examples include ceftriaxone 2 g IV daily, cefotaxime 2 g IV 3 times per day, or ceftazidime 1 g IV every 8 hours. Gentamicin 5 mg/kg IV every 24 hours can be added for *Pseudomonas* coverage, eg, with IV drug users.

The second role of the provider — and probably the reason the patient presented — is the relief of pain. Nonsteroidal anti-inflammatory drugs (NSAIDs), parenteral opioids, and (after arthrocentesis) immobilization are warranted and frequently necessary. All patients determined to have a septic joint should be hospitalized for parenteral antibiotics, drainage as required, and pain control.

There is no evidence upon which to advise the optimal duration of IV or oral antibiotics. Conventionally, they are given IV for up to 2 weeks or until signs improve, then orally for 4 weeks. Symptoms, signs, and acute phase responses are all helpful in guiding the decision to stop antibiotics. Expert review may be required if the expected resolution does not occur.

Weston et al reported that blood cultures were positive in 24% of patients in whom organisms were identified in synovial fluid, and in 9% of these cases a positive blood culture was the only source of a microbiological diagnosis.[44]

Gout

Gout affects 2% of the population of the United States. It is caused by the acute precipitation of uric acid crystals deposited and collected in the joint space. The crystals are ingested by PMN WBCs yielding a cytokine release; this creates an inflammatory synovial reaction, and a painful gout attack ensues. Gout usually presents for the first time in middle-aged men and postmenopausal women, frequently after alcohol intake, purine-rich indulgence (meat, seafood, legumes, dairy, and coffee), illness, trauma (including surgery), and/or new medication.[8,9] Risk factors for gout include obesity, hypertension, diabetes, thiazide diuretic use, cyclosporine use, and lead or radiocontrast exposure.

Patients with a gout attack may appear to be in remarkable pain, perhaps in more discomfort than one who turns out to have a septic joint. The most commonly affected joints are the great toe at the MTP (75%), and the knee, ankle, and tarsal joints. Also, 20% of patients diagnosed with gout will have involvement of more than 1 joint, and they may have bursitis, tenosynovitis, or skin inflammation as well.[45] Patients can have effusions and fever, making it difficult to distinguish gout from septic arthritis based solely on history and physical examination. Importantly, even if a patient indicates a history of gout, this does not rule out infection (cellulitis or

Table 2. Arthrocentesis Interpretation

	Normal	Inflammatory	Septic
Color	Clear/yellow	Yellow/white	Cloudy/opaque
Viscosity	Thick, sticky	NA	Thin, water-like
sWBC	< 25,000/mm^3	NA	> 1100/mm^3 if prosthetic joint > 25,000/mm^3, LR = 2.9 > 50,000/mm^3, LR = 7.7 > 100,000/mm^3, LR = 28
sPMN			> 64% if prosthetic joint > 90%
sLactate	< 5.6 mmol/L	< 5.6 mmol/L	> 5.6 mmol/L, LR = 2.4 to ∞
sLDH	< 250 U/L	< 250 U/L	> 250 U/L
Culture	Negative	Negative	Positive

Abbreviations: LR, likelihood ratio; NA, not applicable; sLactate, synovial lactate; sLDH, synovial lactate dehydrogenase; sPMN, synovial polymorphonuclear; sWBC, synovial white blood cell.

septic joint). Again, as in septic arthritis, serum values and plain films are of limited diagnostic value. **(See Figures 2 and 3.)**

Initially, serum uric acid can be normal or elevated in persons not experiencing a gout crisis. In an acute attack, radiographs may show soft-tissue swelling, while chronic disease will reveal asymmetric bony erosion somewhat removed from the articular surface. Ultimately, it is the finding of intracellular negatively birefringent crystals in the synovial fluid, free of organisms, that clinches the diagnosis of gouty arthritis.

Management Of Gout

Pain control should occur concomitantly with diagnostic work-up. The first-line therapy for this disease is NSAID treatment; in particular, indomethacin, although there are no trials showing indomethacin is more effective in providing pain relief than other NSAIDs.[46,47] Although NSAIDs are traditionally the first-line therapy, there are patients in whom NSAID use is contraindicated, particularly those with true allergy, gastritis, peptic ulcer disease, renal insufficiency, and labile hypertension. In an effort to avert the gastrointestinal toxicity, COX-2 selective inhibitors may be used, but the possible cardiovascular risk may yield more harm than good for this population.

To avert the highlighted risks or side-effects of NSAID use, oral colchicine is a reasonable alternative; however, there have been no direct comparison studies between NSAIDs and colchicine.[48] (Note: Oral colchicine is contraindicated in patients with hepatic and renal insufficiency, and is not removed by dialysis. The parenteral form of colchicine has potential for severe toxicity and should be avoided.[49]) While colchicine, which inhibits microtubule formation and the inflammatory reaction to crystal presence, may be used when NSAID administration is not appropriate, this drug is not without its side-effects. At high dosing (classically, a loading dose of 1.2 mg followed by 0.6 mg/hr for 6 hours; 4.8 mg PO total), acute gout symptoms are relieved, but nausea, vomiting, and diarrhea are frequently experienced and may be severe, making its use undesirable.[48,50] The high-dose regimen is supported by more extensive evidence, but because the newer low-dose regimen (for example, 1.2 mg PO followed by 0.6 mg 1 hour later) has fewer side effects, there is emerging support for this treatment option, which was recently shown to be comparable to high-dose colchicine in a randomized trial of 184 patients.[51] The low-dose regimen is specifically recommended for the elderly.[48]

Also important to consider is the cost of colchicine compared to NSAIDs. Ibuprofen, for example, is relatively inexpensive and may be procured without a prescription. The generic label costs $12.99 for 30 tablets of the 800-mg formulation.[52] In contrast, colchicine is no longer available in generic form, and 30 tablets of the 0.6-mg formulation costs $170.[53] **(See the Cutting Edge And Controversies section, page 15.)** A high cost is generally prohibitive to a significant portion of the target population, so providers should have other options in their armamentarium.

When NSAIDs are contraindicated and colchicine is not an option (either due to adverse reaction or cost), steroids are another affordable and widely available choice. There are no placebo-controlled trials on systemic or intra-articular (IA) steroid use in acute gout flares, but there is a small study that examines the use of a single dose of triamcinolone acetonide 10 mg with good effect.[48,50] Other sources recommend 5 mg to 10 mg triamcinolone for smaller joints, with 20 to 40 mg for knees and shoulders. Pain relief was complete within 48 hours, and no patients reported side-effects or rebound attacks.

In other studies comparing systemic steroids in varying doses and forms to NSAIDs, both agents were found to be equivalent in relief and low side-effect profile.[50] For example, in a recent randomized trial of 90 adult ED patients in Hong Kong with gout-like arthritis, pain scores among those receiving indomethacin/acetaminophen and prednisolone/acetaminophen decreased in a clinically indistinguishable fashion in the ED and in subsequent days (though the prednisolone group ended up consuming twice as much acetaminophen).[54] Systemic steroids may be particularly useful for patients with few other choices and severe gout flares in multiple locations or at sites not easily accessible for local aspiration or infiltration.

Corticotropin is a potential alternative or adjunct to traditional gout therapy. The mechanism of action is poorly understood, but the agent exhibits anti-inflammatory effects. It has multiple potential routes of administration and has been studied with conflicting results. The studies are old and of varying quality, lacking any randomized controlled trials. In comparison to traditional NSAID treatment, administration of corticotropin may result in rapid pain relief, within 2 to 4 hours,[55] or it may take significantly longer, with a mean symptom resolution time of 5 days, in one study.[56] Possible side effects include hypokalemia, worsened glycemic control in diabetics, fluid retention, and gout relapse and flare.[57]

Allopurinol and newer uric acid-lowering agents such as febuxostat, uricase, and pegloticase are useful in the management of chronic gout, in an effort to decrease gout flares. There are no data about the appropriateness of their institution from the ED in the setting of an acute flare, but it is generally recommended that uric acid-lowering agents should not be discontinued if the patient is already on a prophylactic regimen.[58]

In addition to administering drugs for pain relief, providers should consider potential precipi-

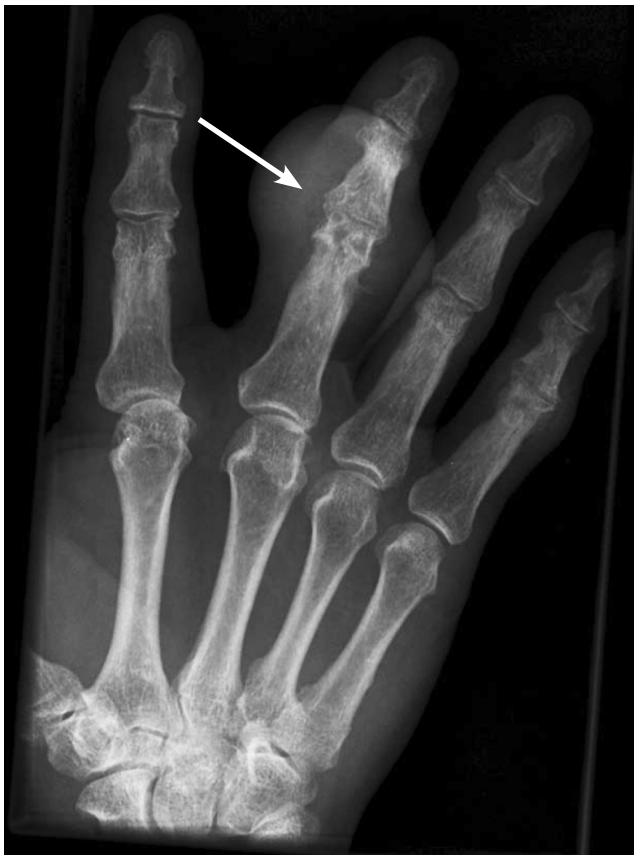
tants. For example, if the patient is on a diuretic, it should be held, and alcohol and dietary counseling should be provided at this teachable moment. Losartan can be started in place of an offending diuretic, as it has modest uricosuric effects.[48] Rest, ice, and elevation can be used in conjunction with medication administration for successful pain relief. Patients should follow up with a rheumatologist to plan suitable prophylaxis and flare treatment options to minimize ED visits. Patients should also be advised to follow up with their primary care providers to determine which medication changes can be safely instituted.

Calcium Pyrophosphate Dihydrate Deposition Disease (Pseudogout)

The pathophysiology of calcium pyrophosphate dihydrate deposition (CPPD) disease, also known as pseudogout, seems to involve chondrocyte overproduction of calcium pyrophosphate and histologic changes to the extracellular matrix of cartilage.[59,60] Prior joint surgery[61] and aging are strongly associated with CPPD, as is hemochromatosis and Wilson disease and endocrine disorders.[48] Most cases are asymptomatic. The true incidence of CPPD is unknown, but when crystals precipitate in the joint space, an inflammatory synovitis (pseudogout) results. The presentation is similar to gout, and it may coexist with gout. Pseudogout tends to afflict patients older than 65 and, most often, tends to involve the knee.[48,62]

The diagnosis of pseudogout is dependent on arthrocentesis and radiography. X-ray demonstration of crystal deposition appearing as punctate, linear densities in articular cartilage is termed chondrocalcinosis and is a feature of CPPD. **(See Figure 4, page 12.)** Synovial fluid examination will show rhomboidal, weakly positive birefringent crystals. The presence of crystals does not preclude septic arthritis; Gram stain and cultures should be obtained.[62]

Figure 2. Tophaceous Gout In A PIP Joint



The arrow points to a gouty tophus and an overhanging margin just proximal to the PIP joint.

Abbreviations: DIP, distal interphalangeal; PIP, proximal interphalangeal.

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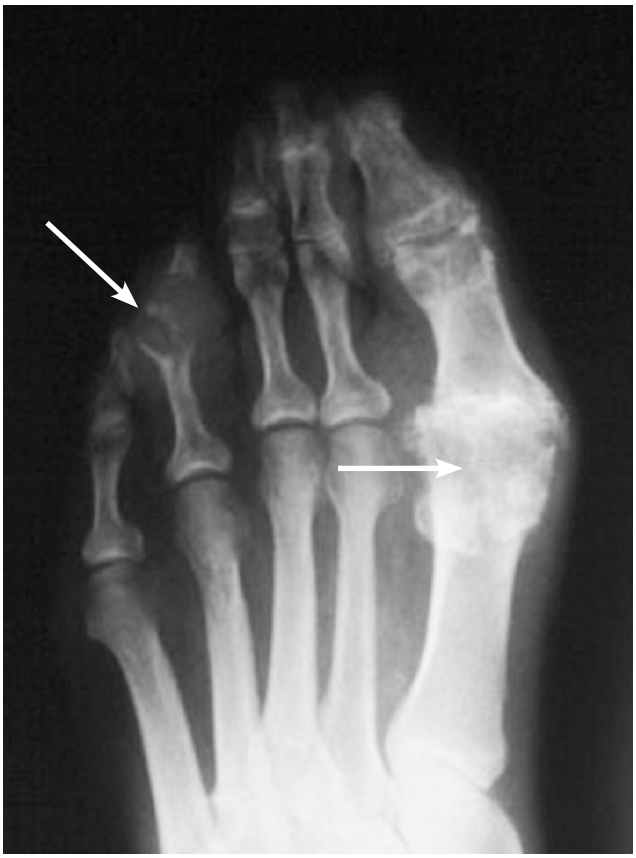
Figure 3. Gout In The First MTP Joint And Fourth DIP Joint

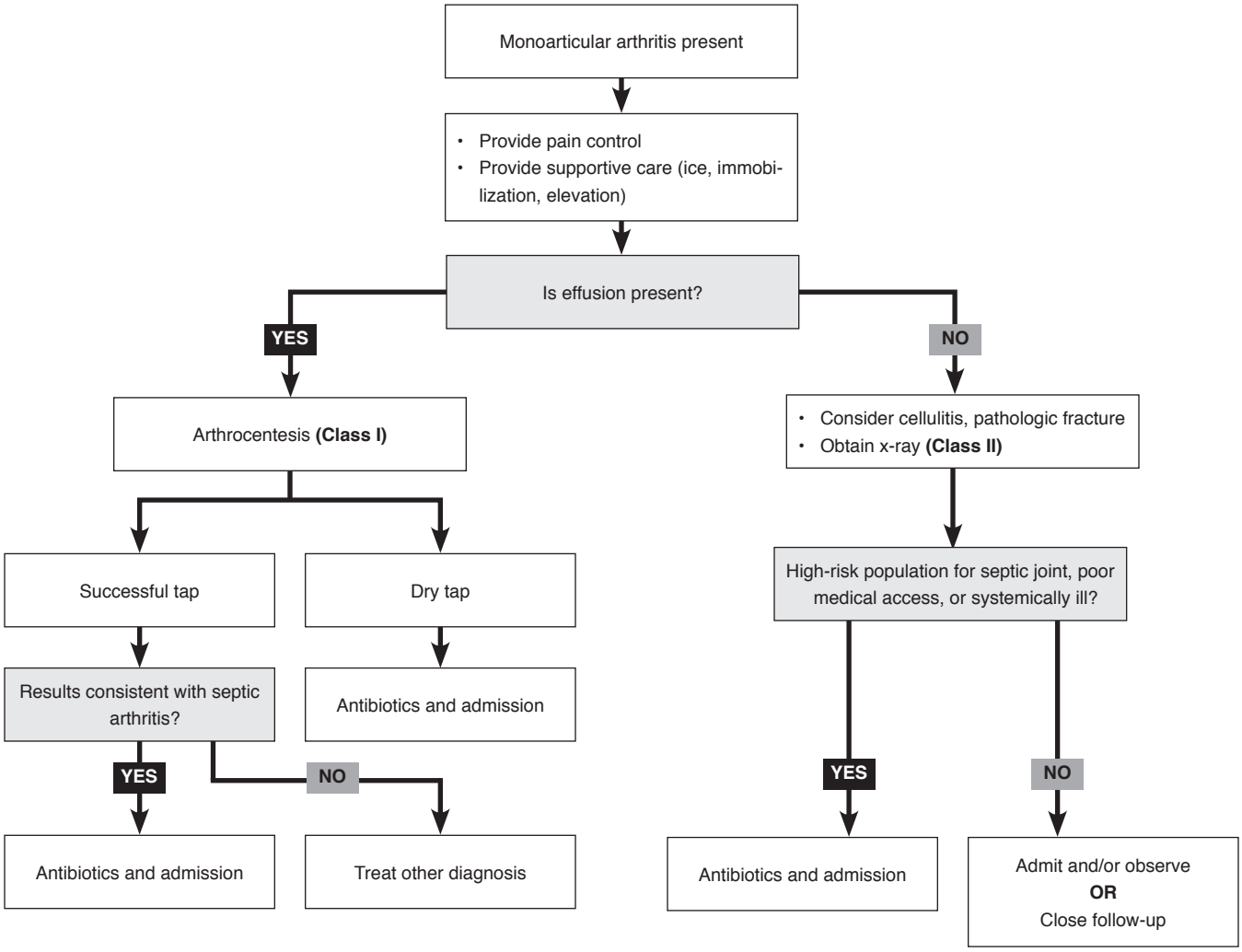


Arrows point to gout in joints of the foot. Note the radiolucent urate depositions and narrow joint space.

Abbreviations: DIP, distal interphalangeal; MTP, metatarsophalangeal.

Image reprinted from radRounds Radiology Network (http://www.radrounds.com) for Academic/Educational purposes.

Clinical Pathway For Workup And Management Of Monoarticular Arthritis



Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA*. 1992;268(16):2289-2295.

Management Of Pseudogout

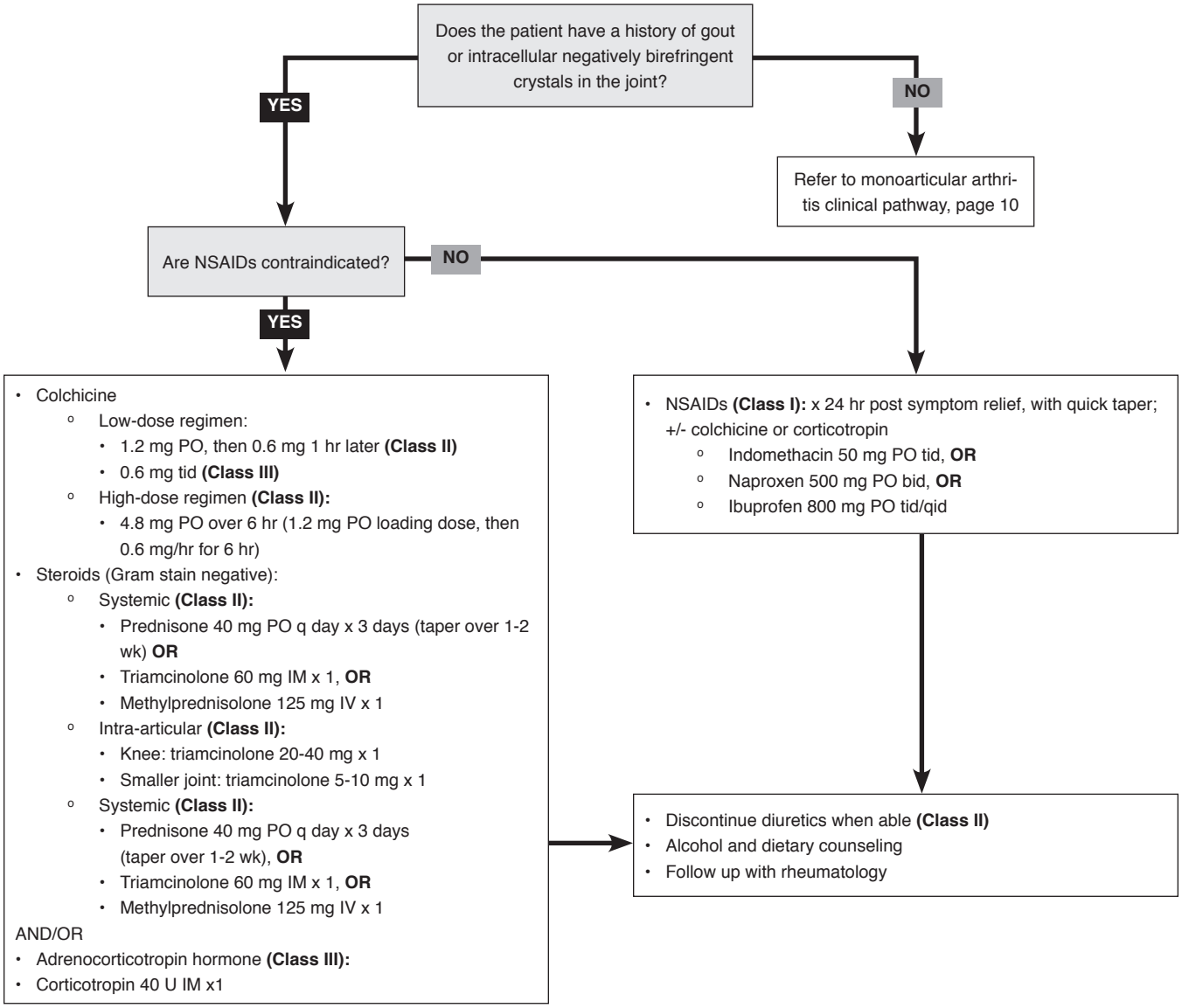
Pseudogout therapy is not supported by randomized trials. Instead, treatments are extrapolated from gout and other arthritis therapies. Recommendations include NSAIDs (with typical precautions for gastrointestinal bleed, renal impairment, and cardiovascular concerns) and colchicine at a dose of 0.5 mg 3 to 4 times per day (a 1-mg loading dose is optional).[63] IA glucocorticoid injections are also recommended by experts, and small trials support intramuscular (IM) and IV steroids over multiday PO steroids.[48]

Osteoarthritis

OA is very common among older Americans. Because cartilage has limited self-repair capability, disturbances in loading (from trauma or excess weight) can lead, over a period of years, to a subchondral bony overgrowth, cartilage degradation, and acutely, synovial membrane inflammation.[64]

Classically, OA pain worsens with activity and improves with rest. In hands, osteophytic spurs in the PIP and DIP (Bouchard and Heberden nodes) may be palpable. Crepitus in other joints, particularly the knee, may be felt and heard on ranging.

Clinical Pathway For Management Of Acute Gout



Abbreviations: bid, 2 times per day; IM, intramuscular; IV, intravenous; NSAID, nonsteroidal anti-inflammatory drug; PO, by mouth; q, every; qid, 4 times per day; tid, 3 times per day.

See page 10 for class of evidence definitions.

Radiographs may show osteophyte formation and subchondral cysts. **(See Figure 5.)** Classically, x-rays will show asymmetrical joint space narrowing, though the patient's symptoms and disability may not correlate with the degree of narrowing on film.

Management Of Osteoarthritis

In the ED, management of OA is geared toward symptom relief. A 2006 Cochrane Review of 15 trials showed that 4 g/day of acetaminophen for hip and knee OA was superior to placebo but inferior to NSAIDs.[65] Because of the risks of NSAIDs in the elderly, acetaminophen is considered first-line treatment in recent ACR and EULAR guidelines. According to a 2007 meta-analysis, opioid dosing in OA has been shown to reduce pain and improve function, though there was substantial heterogeneity and attrition secondary to adverse events.[66] Oral glucosamine and chondroitin, while popular, have shown no clinically significant improvement as reported in a new meta-analysis comparing their use, alone or in combination with other drugs, to placebo.[67]

Topical analgesics such as capsaicin and diclofenac have shown promise in prospective trials in nonemergency settings.[68,69] A meta-analysis of 4 randomized trials of topical diclofenac (1.0%-1.5%) showed superiority over placebo and better tolerance, with similar efficacy, compared to the same agent given by mouth.[69] Side effects to topical diclofenac tend to occur over long-term use and primarily involve local skin reactions.

IA injections of glucocorticoids (such as triamcinolone 10 mg for small joints, 20 mg for elbows and ankles, and 40 mg for hips, knees, and shoulders) has demonstrated months of pain relief in randomized trials, though no change to physical function, and efficacy varied in different joints.[71]

Compared to IA steroids, IA hyaluronic acid has shown superior pain relief and improvement in function, according to 2 recent studies.[71,72] Improvement occurred only after a period of days to weeks, however, and patients must be advised of the expected delay in benefit. Prior to these 2 studies, a meta-analysis concluded that there was no benefit and that there was, possibly, a higher risk of adverse events with IA hyaluronic acid.[73]

IA lidocaine and bupivacaine have been studied in the context of ED joint reduction and postoperative pain management, but there are no studies to support their use for OA analgesia in the ED, alone or in combination with other agents, and there are new concerns regarding chondrotoxicity. **(See the Cutting Edge And Controversies section, page 15.)**

Other Monoarthritides

While traditionally classified as "polyarticular," many arthritides can manifest with monoarticular complaints, particularly Lyme arthritis and reactive arthritis (formerly called Reiter syndrome). Other possibilities, such as Still disease, relapsing polychondritis, psoriatic arthritis, enteropathic arthritis, and fibromyalgia are beyond the scope of this review.

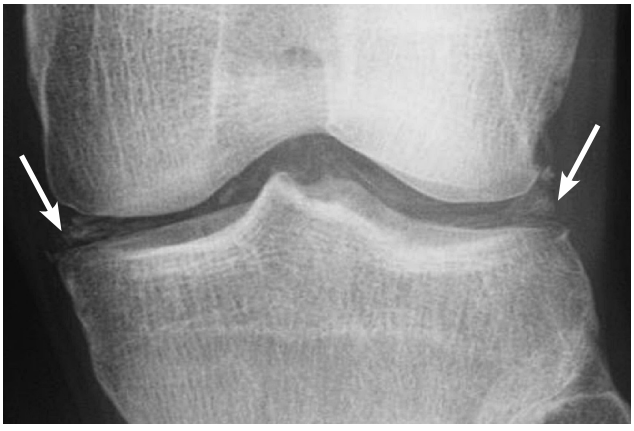
Lyme Arthritis

After an initial period of myalgias and arthralgias in the early disseminated phase of Lyme disease, 50% to 60% of Lyme disease patients will progress to a frank asymmetric arthritis within 6 months. This arthritis is the most common manifestation of late-phase Lyme disease and appears to be mediated by an autoimmune process, rather than direct infection.[11,74] Clinically, patients with Lyme arthritis often present with a large effusion, commonly in the knee, though fevers are unusual and joint pain is minimal.[75] The patient may not remember or may not have experienced the classic erythema migrans rash or tick bite. Synovial fluid analysis will show an inflammatory state with PMN predominance, though cultures will be negative for *Borrelia burgdorferi.* Confirmation depends upon immunoglobulin M and G serology. Guidelines for Lyme arthritis recommend a 4-week course of doxycycline (100 mg 2 times per day),[75] though amoxicillin 500 mg 3 times per day or cefuroxime 500 mg 2 times per day are acceptable alternatives. IV antibiotics are reserved for refractory arthritis or if there are concomitant neurologic or cardiac symptoms of Lyme disease.

Reactive Arthritis

Reactive arthritis is a rheumatoid factor (Rf)-negative spondyloarthropathy resulting from infection from *Chlamydia trachomatis* in the genitourinary tract, or gastrointestinal infection of *Shigella, Salmo-*

Figure 4. Articular Cartilage Chondrocalcinosis



Arrows point to linear, punctate calcifications in the knee. This chronic feature of calcium pyrophosphate dihydrate deposition can be seen in an acute flare (pseudogout) in the emergency department.
Image reprinted from radRounds Radiology Network (http://www.radrounds.com) for Academic/Educational purposes.

nella, Campylobacter, or other agents.[76,77] Arthritis symptoms appear 2 to 6 weeks after an episode of dysentery or urethritis (or, in women, a cervicitis, potentially asymptomatic). Oral and genital lesions or conjunctivitis may be seen in a small fraction of patients, though these findings often pass before the onset of arthritis. Patients are generally between ages 20 and 40 and have asymmetric, often polyarticular involvement of lower-extremity weight-bearing joints, particularly the heel. Episodes may last for months, may remit and recur, or may progress steadily. As with Lyme arthritis, the synovial fluid is inflammatory, with predominantly sPMNs. Cultures are sterile. NSAIDs, particularly indomethacin 50 mg 3 to 4 times per day, are recommended.[76] In patients with reactive arthritis triggered by *Chlamydia,* antibiotics shorten the duration of symptoms, though this is not true of arthritis triggered by gastrointestinal pathogens.

(Note: Reactive arthritis was first described by Hans Reiter, a Nazi physician who was found guilty of war crimes for inhumane experiments in concentration camps during World War II. The physician who coined the term "Reiter syndrome" in 1942 has successfully advocated for "reactive arthritis" to replace the eponymous term, and major rheumatology societies have agreed.[78])

Traumatic Arthritis

Trauma is a common cause of an acute monoarticular effusion, often as the result of ligamentous injury, but it is also associated with IA fractures. In knee trauma, one prospective series of tense hemarthrosis within 12 hours of injury identified complete or partial anterior cruciate ligament (ACL) tears in 70% of patients and meniscal tears in 16%. Synovial disruption was presumed responsible in 5%.[79] An older, prospective series of 100 acute trauma patients with knee hemarthrosis in the United Kingdom found that despite normal radiographs and confirmation of hemarthrosis by aspiration, 99 patients had serious pathology such as ACL, posterior cruciate ligament (PCL), or collateral ligament tears seen with prompt arthroscopy.[80] In both studies, authors recommended aspiration of a tense effusion and urgent arthroscopy.[79,80] Certainly, unstable weight-bearing joints and IA fractures should be evaluated in the ED by orthopedists; otherwise, traumatic hemarthrosis can be managed with immobilization, elevation, ice, and pain medication, with outpatient orthopedic follow-up.

Controversies And Cutting Edge

Computed Tomographic Evaluation Of Gout

Because pathologic findings from gout may take years to manifest with x-ray findings, and because crystals may not be seen in a fraction of joint aspirations (and because, we suspect, aspirations of small gouty joints is a daunting prospect), noninvasive means of diagnosing gout have been sought.[81] A new protocol for dual-source, dual-energy computed tomography (CT) using a mineral composition algorithm has shown promise in a small trial diagnosing uric acid collections in joints. Blinded radiologists were able to match 12 positive CTs to 12 positive joint aspirations for gout; specificity was also good.[81] This protocol for CT analysis of gout is not widely available, nor has the role of CT for diagnosis of gout in the ED been prospectively evaluated.

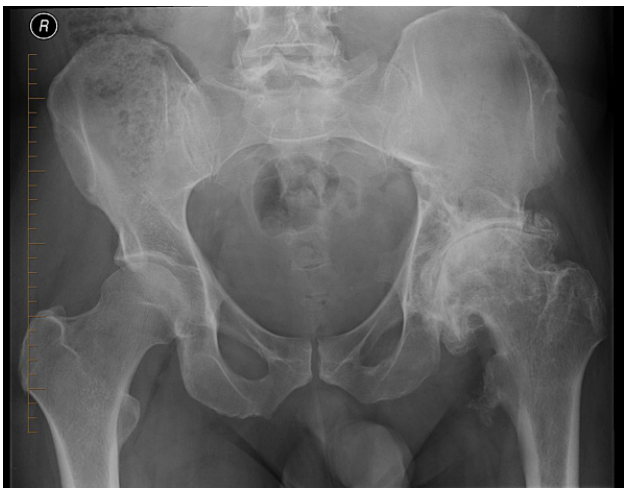
Ultrasound Evaluation Of Gout

Because crystalline material in gouty joints reflects ultrasound waves more strongly than soft tissue and cartilage, ultrasound can theoretically be used to identify crystalline arthropathies such as gout or CPPD. A "double-contour" sign, produced by the deposition of urate crystals on articular cartilage, has been associated strongly with gout.[19] **(See Figure 6, page 15.)**

Colchicine Pricing

Colchicine has been used to treat gout since ancient times, but its price in the United States has risen dramatically in recent years. It's no coincidence that suggested dosing regimens have changed in recent years, as well. The reason is that the United States Food and Drug Administration (FDA), which had never formally reviewed colchicine, as its use was grandfathered in as an approved therapy by the FDA at its inception in 1938. Authors affiliated with URL Pharma, Inc. conducted a trial that showed

Figure 5. A Normal Hip And An Osteoarthritic Hip



Compared to the normal hip (seen on the left), the osteoarthritic hip (seen on the right) shows narrowed joint space, osteophytes, and sclerosis (increased bone density at the articular surfaces).
Image used courtesy of Dr. Frank Gaillard, www.radiopaedia.org, Creative Commons BY-SA-NC.

superior efficacy and somewhat improved safety of a simplified lower dosing of colchicine (1.2 mg followed by 0.6 mg an hour later) compared to existing high-dose regimens.[47]

These results were submitted to the FDA and, in 2009, under the exclusivity provisions of Hatch-Waxman, URL Pharma, Inc. was granted 3 years of market exclusivity for what was considered a new indication for colchicine – the safe treatment of gout. The price of colchicine (now sold under the name Colcrys®) has risen from $0.09 per pill to around $5.67 per pill.[53] Despite the ire of some lawmakers and journal editors, this state of affairs is consistent with the letter of the law (though its intent must have been to produce a greater benefit to patients than this). The exclusivity period should end in August 2012, though it is not clear if other manufacturers of colchicine will be able to readily re-enter the market.

Risk Management Pitfalls For Monoarticular Arthritis

1. **"When I consulted the orthopedist about the hot, red, swollen knee, she asked about the ESR and CRP. So I'm waiting on those before I do the tap."**
   There is no serum blood test that can rule out septic arthritis.

2. **"The red, hot, swollen ankle may have been an overlying cellulitis, so I didn't do the tap."**
   While there is a theoretical risk that aspirating through cellulitis may inoculate a sterile joint with bacteria, there is a more concrete, measurable risk in skipping arthrocentesis and prompt management in a septic joint. Try the tap, through cellulitic-looking tissue, if necessary.

3. **"That elderly lady with a history of RA and a recent COPD flare didn't have a fever or too much unilateral joint pain and swelling – there's no way that could be septic arthritis."**
   Elderly patients may not present with an impressive examination, but they are still at risk for septic arthritis, especially if they're immunocompromised (eg, on steroids for COPD) or if they have a history of RA.

4. **"My patient with knee OA keeps coming to the ED, asking for pain meds. So what else am I supposed to do?"**
   Nonpharmacologic therapy such as orthotic shoes, canes, splints, and immobilizers have been shown to help in OA – so does specialty follow-up. Try it.

5. **"That lady with gout responded really well to NSAIDs – so I'll discharge her with a script for more."**
   Gout goes hand in hand with renal insufficiency – and other therapies are less nephrotoxic. Investigate further or try another therapy before writing a script for NSAIDs.

6. **"Look, this joint seems septic. I sent the synovial fluid and I'm calling ortho – I don't have time to ask about sexual history."**
   Gonococcal arthritis is managed nonoperatively, and HIV can cause arthritis in several ways that won't resolve at the time of hospital discharge. Investigate further.

7. **"My patient with gout had a long laundry list of meds and had a bunch of questions about diet – I'll just treat the pain and let the primary care doctor sort it out."**
   In gouty flares, temporarily withholding thiazide diuretics and abstaining from alcohol and purine-rich foods will probably help decrease the duration and intensity of symptoms.

8. **"My patient had classic x-ray findings of gout (or pseudogout). So what if there was a fever and the joint had never looked so red, hot, and swollen in prior flares?"**
   Septic arthritis can coexist with gout or pseudogout – sending Gram stain and culture can save the joint and the patient.

9. **"It's odd this teenager has a huge swollen knee, but it's not that painful, and he doesn't recall a tick bite or rash. It can't be Lyme."**
   Patients with Lyme arthritis may not recall a tick bite or may not have experienced erythema migrans. Treat based on exposure and clinical presentation.

10. **"The joint wasn't uniformly red and hot, but there was an area with swelling, and the patient said it really hurt a lot with flexion."**
    Bursitis and tendonitis can look like septic arthritis – but in septic arthritis, the joint should hurt with any active or passive ranging. Ask about minor trauma and repetitive stress.

Intra-articular Analgesics

It has been common practice to instill amide analgesics such as lidocaine or bupivacaine intra-articularly, either alone or mixed with steroids, hyaluronic acid, or other agents. Recently, however, in vitro and now in vivo animal models suggest even a single IA dose of bupivacaine can show long-lasting chondrotoxicity.[23] Rats injected with 0.5% bupivacaine at their knees had up to 50% depletion in chondrocyte density at 6 months, compared to IA saline injections. Histologic abnormalities were apparent at 4 weeks, though only below the articular surface. The implications for clinical practice from in vitro and in vivo work are not clear, as chondrocyte depletion in people may take years or decades to lead to clinically significant cartilage degradation, if at all.

Nonetheless, reports are emerging of chondrolysis after several days of bupivacaine exposure in the setting of arthroscopy pain management. One retrospective review showed that 19% of patients (3 of 16) with glenohumeral joint infusions of 0.5% bupivacaine pain pumps developed severe cartilage degeneration within 12 months.[82] Another study looked at the use of IA amide analgesics after arthroscopic surgery and found mild, but significant, superiority over placebo.[22] The authors concluded that IA bupivacaine and similar agents – while slightly more effective – are not worth the risk of chondrotoxicity, so they recommend systemic pain medications instead. While harm from a single properly administered IA bupivacaine injection in an ED patient complaining of joint pain has not been reported, the balance of in vitro, animal, and postoperative evidence seems to be suggesting that risk of cartilage damage is possible.

Point-Of-Care Synovial Lactate

In a recent review of 4 trials spanning decades, synovial lactate > 10 mmol/L has been shown to increase the likelihood of septic arthritis dramatically (+LR ranged from 2.4 to infinite in 2 trials, meaning perfect correlation in those limited samples between meeting the synovial lactate threshold and having septic arthritis).[24] These trials measured lactate on laboratory chromatographs or spectrophotometers. Modern point-of-care testing has not been studied for accuracy in measuring synovial lactate for diagnosing septic arthritis; however, serum lactate measurements at the point of care have proven reliable in diagnosing sepsis.[83,84] Point-of-care synovial lactate measurements may soon be a validated and rapid way to diagnose septic arthritis in the ED.

Special Populations

Patients With Prosthetic Joints

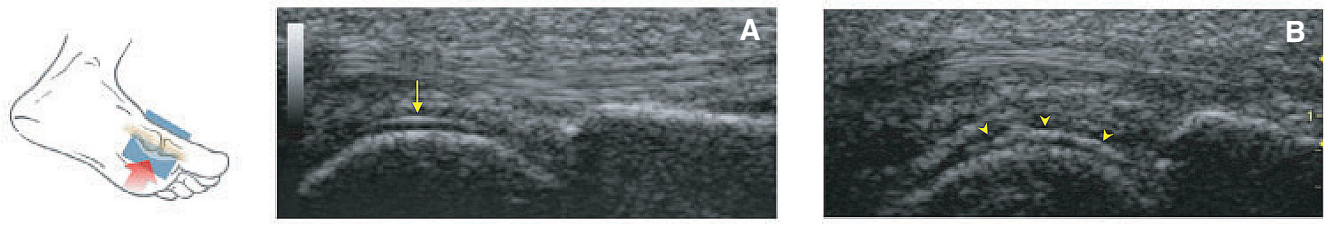
Prosthetic joints are at a substantially higher risk of septic arthritis than native joints. Host immune response and antibiotic bioavailability are decreased in the prosthetic environment. Infections in prosthetic joints are classified as "early" when they occur within a month of surgery, or "late" when they occur more than a month after surgery. Late infections are often the result of hematogenous spread from other infections, though indolent organisms introduced at the time of surgery have been shown to surface up to a year later.[85]

Many organisms have been identified from prosthetic joint infections.[86] Arthrocentesis is best performed in consultation with the operating surgeon, due to postsurgical changes in anatomy that might render the procedure more difficult as well as a perceived higher risk of introducing infection, including osteomyelitis.[16] Synovial marker thresholds for diagnosing septic arthritis are lower in the case of prosthetic joints; prior studies suggest that an sWBC count of > 1100/mm^3 or an sPMN concentration of > 64% is sensitive and specific for septic arthritis.[41]

Patients With Human Immunodeficiency Virus

While the overall risk of rheumatologic complications in HIV remains low, ED providers should be aware of several distinct etiologies for arthritis or periarticular symptoms. Most straightforward

Figure 6. Ultrasound Of MTP Joints, With And Without Gout



Plantar longitudinal ultrasound shows, in view A, a healthy MTP joint with a black rim of hyaline cartilage and a reflex artifact at the apex. In a patient with gout (view B), the hyaline cartilage is covered with a thicker, irregular band.

Abbreviation: MTP, metatarsophalangeal.

From: R. G. Thiele and N. Schlesinger. Diagnosis of gout by ultrasound. *Rheumatology.* 2007;46(7):1119. By permission of Oxford University Press.

is opportunistic septic arthritis in patients with acquired immune deficiency syndrome (AIDS). In the era before highly active antiretroviral therapy (HAART), patients with HIV were also observed to develop largely monoarticular arthritis, often involving the feet and ankles, that bore a resemblance to reactive arthritis or psoriatic arthritis. Additionally, an atypical and difficult-to-categorize arthritis was observed, thought to be related to direct HIV infection of synovium.[41]

Since the advent of HAART, the incidence of those forms of arthritis in HIV has declined; however, HAART itself is said to be responsible for a self-limited immune reconstitution syndrome. This syndrome mimics RA, or activates a previously unrecognized RA in patients. Rather than discontinue HAART, specialists try to strike a balance between the lowest possible glucocorticoid immunosuppressants and continued antiretroviral therapy.[41,87]

Disposition

The disposition of the patient with monoarticular arthritis revolves around the diagnosis: if septic arthritis is not satisfactorily excluded by examination and ED workup, then the patient should be on antibiotics and admitted to the hospital. Admission is reasonable in cases of refractory pain or for concerns about patient safety or inadequate follow-up. Other cases of monoarticular arthritis can be managed as outpatients, provided that the patient's pain is controlled, can perform activities of daily living safely, and will follow up reliably.

Specialty follow-up has been studied with respect to gout management and is recommended for new diagnoses of gout as well as refractory cases and for patients with complex needs.[88] Beyond pharmacologic pain relief, the emergency clinician would do well to recall the time-honored and often evidence-based therapies of orthotics, splints, immobilizers, and canes, which have merit for some cases of OA and RA and likely play a role in care for more acute monoarticular arthritides.

Summary

Monoarticular arthritis presentations in the ED must prompt a consideration of septic arthritis unless the history overwhelmingly favors an alternative diagnosis (such as a recent trauma or long course of pain in the affected joint, with gradual flare). Even a history of gout in the same joint should give pause before dismissing septic arthritis. In a new, atraumatic, hot, swollen joint, there is no combination of examination findings and blood testing that puts the patient below the threshold for arthrocentesis. Furthermore, bacterial arthritis can coexist with gout or pseudogout, and Gram stain is not always positive in cases of septic arthritis. Cultures should be sent. Once septic arthritis is excluded, NSAIDs or acetaminophen are the first-line recommended therapy for gout, OA, and other common arthritides. If contraindications to NSAIDs

Time- and Cost-Effective Strategies For Monoarticular Arthritis

- Don't wait for blood test results before arthrocentesis. While consultants may recommend serum ESR, CRP, or WBC levels, there is no blood test that can safely put septic arthritis below the threshold for arthrocentesis.
- Don't trust that the laboratory knows what to do with your sample. Hoping the laboratory will know what to do with your checkboxes and synovial fluid sample is risky, especially if there is not much in the syringe. Call them as soon as you send the sample and tell them what pathologies — from gonococcal arthritis to pseudogout — you're concerned with.
- Don't wait for films. While x-rays may be helpful in establishing a baseline for joint disease and in ruling out osteomyelitis or fracture, there is nothing in plain films — such as tophi, joint space narrowing, or chondrocalcinosis — that could convince you to forego arthrocentesis for suspected septic arthritis.
- Don't wait for results of arthrocentesis to start pain medication. While it may be prudent to withhold antibiotics in ambiguous cases of septic arthritis, patients with gout or pseudogout are in a lot of pain, and they can be treated empirically with medications like NSAIDs, if safe, while waiting for crystal analysis.
- Dose colchicine effectively. The traditional regimen of 0.6 mg every hour until gastrointestinal symptoms arise is neither safe nor efficient. The FDA-approved new regimen is 1.2 mg followed by 0.6 mg 1 hour later – and that's it. Consider other adjuncts (NSAIDs, steroids) for greater pain relief.
- Try a point-of-care sLactate. Studies show that sLactate levels > 10 mmol/L rule in septic arthritis. While point-of-care lactate testing for septic arthritis has not been formally evaluated, point-of-care lactate testing in sepsis has proven accurate, reliable, and much faster than laboratory analysis.

are present or pain has not been sufficiently relieved, many arthritides can be relieved with steroids (systemically or IA dosing). Appropriate follow-up with orthopedics or rheumatology is important for optimal management and future flare-up prophylaxis.

Case Conclusions

You remembered from your evidence-based review of the literature that there is no serum blood test that can adequately rule out septic arthritis, so the patient's history and exam warranted arthrocentesis. After laying the patient flat and partially flexing the knee with a pillow, you guided the needle medially under the patella and you aspirated watery, but cloudy, material. A point-of-care sLactate came back quickly at 15 mmol/L, and removed any ambiguity — this was a septic joint. While synovial culture and Gram stain (and blood cultures) were sent, along with sWBC and pre-op labs, you initiated IV antibiotics — vancomycin and ceftriaxone. Then you called up the orthopedist and asked him to prepare the OR.

As for your other patient with shoulder pain, x-rays confirmed degenerative joint disease without fracture or calcific tendinitis. Since (based on history and exam) you did not suspect septic arthritis, you set about the task of relieving his pain. As the patient predicted, NSAIDs were no longer doing much to help him. You dosed a few tablets of acetaminophen/oxycodone and prepared an IA injection. A few years ago, you might have reached for 40 mg triamcinolone mixed with 5 mL of 0.5% bupivacaine, but now you're aware of the theoretical risk of cartilage damage from IA analgesics. Then, after sterile prep, you followed your landmarks and injected the triamcinolone posteriorly into the shoulder, applied topical diclofenac to the shoulder, and wrote a prescription for the same.

This Month In *EM Practice Guidelines Update*

The May 2012 issue of *EM Practice Guidelines Update* reviews 2 guidelines that focus on the management of atrial fibrillation in the ED: (1) The Canadian Cardiovascular Society Atrial Fibrillation Guidelines 2010: Management of Recent-Onset Atrial Fibrillation and Flutter in the ED, and (2) 2011 ACCF/AHA/HRS Focused Update on the Management of Patients With Atrial Fibrillation. Subscribers to *Emergency Medicine Practice* have free access to this online publication at www.ebmedicine.net/AFib

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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1. Which form of arthritis is most common among adults in the United States?
   a. Septic arthritis
   b. OA
   c. Gonococcal arthritis
   d. Gout

2. A man presents with a hot, red, swollen knee. Which of the following findings make septic arthritis more likely?
   a. His knee x-ray shows an overhanging margin
   b. He is 40 years old
   c. He has never had knee pain or surgery, or a diagnosis of arthritis
   d. His knee only hurts when flexed

3. A woman presents with a hot, red, swollen ankle. Which of the following comfortably rules out septic arthritis?
   a. She has had gout in this ankle before
   b. She has no fever
   c. She has an ESR of 40 mm/hr
   d. She has an sWBC count of 5000 cells/mm^3

4. You aspirate a hot, red, swollen joint because you're concerned for septic arthritis. Which of the following findings from joint fluid aspiration makes septic arthritis more likely?
   a. Viscous aspirate
   b. Synovial LDH of 300 U/L
   c. Synovial lactate of 3.0 mmol/L
   d. Synovial WBC count of 15,000 cells/mm^3

5. Which of the following regimens for antibiotics is most appropriate in an IV drug user with a septic joint?
   a. First-generation cephalosporin and vancomycin
   b. Third-generation cephalosporin and vancomycin
   c. Ceftazidime and gentamicin
   d. Ampicillin and sulbactam

6. **Which of the following therapies has NOT been shown to be helpful in managing knee OA?**
   a. Orthotic shoes
   b. Weight loss
   c. Acetaminophen
   d. Chondroitin

7. **Which of the following is NOT true of Lyme arthritis and reactive arthritis?**
   a. Both are typically polyarticular but can present as monoarticular arthritis
   b. When polyarticular, both are typically asymmetrical
   c. Both are exquisitely painful
   d. Both are associated with infection weeks before arthritis symptoms

8. **A man presents with weeks of painless oral lesions and a painful left heel. Today, he noticed his right knee has become painful and swollen. Last month, he had burning urination. A medication shown to shorten the duration of symptoms in this condition is:**
   a. Prednisone
   b. Azithromycin
   c. IA hyaluronic acid
   d. Oxycodone

9. **The FDA recently approved colchicine for use with acute gout in the following dosing regimen:**
   a. 0.6 mg PO every hour until gastrointestinal symptoms develop
   b. 0.6 mg PO 3 times a day
   c. 1.2 mg PO, then 0.6 mg PO an hour later
   d. 1.2 mg IV

10. **Which of the following is NOT a reason a patient with advanced HIV could develop monoarticular arthritis?**
   a. Direct HIV infection of synovium
   b. Reaction to antiretroviral medications
   c. Opportunistic septic arthritis
   d. Calcium complex deposition

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